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Long-term cardiovascular events in individuals hospitalised with COVID-19: a retrospective cohort

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Abstract

Background Post-COVID condition encompasses a spectrum of persistent or emerging symptoms affecting multiple organ systems, including a heightened risk of cardiovascular complications. Despite growing recognition of this phenomenon, there remains a lack of comprehensive data regarding the incidence and risk factors associated with cardiovascular events during the post-acute phase in patients previously hospitalised for COVID-19. Thus, we aimed to estimate the incidence of cardiovascular events among patients hospitalised for COVID-19 in Portugal and assess the association between patient and infection characteristics and cardiovascular events in the COVID-19 post-acute phase.

Methods We conducted a registry-based retrospective cohort study from seven hospitals across Portugal. Data was retrospectively collected from the electronic medical record of each patient. We included individuals hospitalised due to COVID-19 between March 2020 and March 2021. Our outcome of interest was cardiovascular events in the post-acute phase of COVID-19, occurring at least 30 days after a positive SARS-CoV-2 test. The variables of interest considered were the severity of the episode, existing cardiovascular risk and vaccination status before the SARS-CoV-2 test. Person-years was estimated for each individual, and incidence rates were estimated. A Cox proportional hazard regression model was employed to assess risk factors.

Results We included 1,803 patients in the analysis, of which 143 (7.9%) experienced at least one cardiovascular event following COVID-19 hospitalisation. The overall incidence rate of having at least one cardiovascular event was 34.65 per 1,000 person-years (95% confidence interval (CI): 29.20; 40.82). We found higher risk of cardiovascular events for individuals with pre-existing cardiovascular risk (adjusted hazard ratio (aHR): 3.76, 95% CI: 1.53; 9.24) and lower risk for individuals with at least one vaccine dose before the SARS-CoV-2 test (partial vaccination – aHR: 0.44, 95%CI: 0.30; 0.64, complete vaccination – aHR: 0.46, 95%CI: 0.2; 0.80). We did not find a significant difference between the severity of the COVID-19 episode and the risk of having cardiovascular events post-COVID-19.

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Conclusion Our findings suggest a substantial burden of cardiovascular complications post-COVID-19, underscoring the need for health services to be prepared and commence screening and preventive measures for individuals at higher risk.

Keywords Post-acute COVID-19 syndrome, Cardiovascular diseases, COVID-19 vaccines

Background

Since the World Health Organization (WHO) declared coronavirus disease 2019 (COVID-19) a pandemic in March 2020, over 770 million cases and 7 million deaths have occurred globally [1]. Among recovered patients, 10 to 70% develop Long COVID or Post-COVID Condition (PCC) [2–6], characterised by persistent or emerging symptoms affecting multiple systems, with symptom onset varying between one to three months [7–9]. These symptoms include fatigue, breathing difficulties, and cognitive issues [10–13] and can last for months, impacting up to 70% of hospitalised cases. However, these prevalence estimates decrease when considering only vaccinated individuals (10% to 30%) [4, 14, 15]. Cardiovascular symptoms persist in approximately 25% of COVID-19 survivors, enduring for months following the initial acute infection [16].

Several studies have shown that beyond the acute infection stage, individuals who have COVID-19 are at an increased risk of developing cardiovascular diseases, including cerebrovascular disorders, dysrhythmias, ischemic and non-ischemic heart disease (pericarditis, myocarditis, and heart failure) and thromboembolic disease, as well as respiratory function abnormalities [17–21]. Despite the number of studies into the prevalence of cardiovascular diseases in the post-acute phase following COVID-19 infection, consensus and standardised data remain lacking across studies.

Arrhythmias are among the most frequently observed complications, ranging between 8 and 27% [22, 23]. Additionally, other cardiovascular complications such as thromboembolism (1.1% to 2.4%), heart failure (1.18% to 2%), myocarditis, and stroke (both less than 1%) have been reported to manifest months after the acute phase [23–25]. While the prevalence of these complications may appear relatively low, it is crucial to acknowledge potential underestimations. Small-scale population studies utilising magnetic resonance imaging (MRI) diagnostics reveal that 6% to 7% of COVID-19 patients exhibit myocarditis-like patterns, and 13% to 19% display indications of infarction within 28 to 68 days post-infection [26, 27]. Moreover, Roca-Fernandez et al. [20] found that one in five individuals experiencing PCC symptoms exhibit abnormalities in MRI scans six months after initial COVID-19 symptoms, with over half still showing abnormalities 12 months later. Another study with 13,435 adults with PCC revealed significantly increased demand for healthcare utilisation associated with cardiac arrhythmias, pulmonary embolism, ischemic stroke, coronary artery disease, and heart failure one-year post-acute infection [21]. Together, these findings suggest a plausible scenario of underdiagnosed cardiovascular complications.

Despite the risk of cardiovascular diseases after COVID-19 being recognised, the underlying factors remain poorly understood. Evidence suggests that the severity of the acute infection, particularly requiring intensive care unit (ICU) admission and lack of vaccination, are linked to an increased risk of post-COVID cardiovascular diseases [10, 18, 22]. Furthermore, pre-existing conditions such as diabetes, hypertension, and other cardiovascular diseases are associated with a higher likelihood of severe acute infection, underscoring the importance of investigating these factors as potential contributors to the long-term development or worsening of cardiovascular diseases following COVID-19 infection [28, 29].

Future research remains crucial to understand cardiovascular issues post-COVID-19 infection. Thus, we performed a registry-based retrospective cohort study to estimate the incidence of post-COVID-19 cardiovascular events among patients hospitalised due to COVID-19 between March 2020 and March 2021 in Portugal. We also assessed the association between patient and infection characteristics and cardiovascular events 30 days after the SARS-CoV-2 test.

Methods

Study design and population

We assessed the electronic registries of seven Portuguese hospitals: Póvoa de Varzim/Vila do Conde Hospital Centre and Vila Real Hospital (North), Coimbra University Hospital Centre and Tondela-Viseu Hospital (Centre), Cascais Hospital (Lisbon and Tagus Valley), Algarve University Hospital Centre – Portimão Unit (Algarve), and Funchal Central Hospital (Autonomous Region of Madeira). Individuals included in the analysis were mainly identified using the International Classification of Diseases (ICD)–10 codes for COVID-19 attributed to each admission episode between March 2020 and March 2021. However, when specialised teams codify events a posteriori, hospitals might experience codification delays. Thus, in those hospitals, a list of patients admitted to special COVID-19 units was requested from the informatics department and used to enrol the patient in the study. We excluded individuals who died during hospitalisation,

with COVID-19 but were admitted due to another cause (e.g. pregnant women, emergency surgery), who did not live in Portugal, and who died or were lost to follow-up within the first 30 days after the SARS-CoV-2 test. Data was retrospectively collected from the electronic medical records of each patient by the hospitals' medical staff (indirect data collection) in a fully anonymised form created for this study. Basically, medical staff were responsible for recording any risk factors, the severity of the COVID-19 hospitalisation, any events of interest, and vaccinations as they have access to patients clinical records and can access and review them to identify any of the events of interest. Medical staff have access to the entire patient records, including any admission to the emergency department, hospitalisations, and follow-up hospital and primary care appointments. Only confirmed diagnosed events of interest were registered for the study. Data extraction was tested with hospital coordinators, and each professional involved in data collection received training to reduce possible biases. Medical staff were involved in data collection, whereas researchers were responsible for the analysis. To maintain the patient anonymity, an identifier was created for each hospital, which was unknown to researchers.

Additional information about the study design can be seen in the published protocol [30].

Variables

Our primary outcome of interest corresponds to the presence of any cardiovascular event, occurring at least 30 days after the positive SARS-CoV-2 test. These were defined as any of the following events after a COVID-19 diagnosis: heart failure, myocarditis, arrhythmia (including cases of atrial fibrillation, atrial flutter, paroxysmal supraventricular tachycardia, ventricular tachycardia or ventricular fibrillation), acute myocardial infarction (with and without ST-elevation), pulmonary thromboembolism, ischemic stroke, hemorrhagic stroke, intracardiac thrombus and acute deep vein thrombosis. An additional field was provided to manually insert any other event (cardiovascular, neurological or respiratory) that might be of interest. This field was reviewed, and events were grouped into different categories whenever applicable and possible.

The medical staff also extracted information on comorbidities before hospitalisation due to COVID-19, such as duration, medication administered, need for ventilation, and, when available, lifestyle behaviours concerning alcohol use, smoking, and physical exercise. The date of the last contact with the services was registered as the number of days since a positive RT-PCR test for SARS-CoV-2 to calculate the follow-up time.

The severity of the COVID-19 episode was classified based on WHO guidelines: mild disease corresponded to patients without supplementary oxygen, moderate disease to patients with supplementary oxygen during admission, and severe disease to patients who needed ventilation or were admitted to the ICU [31]. We also created a variable indicating whether the participant had pre-existing cardiovascular risk. This variable was created considering history of cardiovascular disease corresponding to any cardiovascular event reported before the date of the SARS-CoV-2 test (ischemic/hemorrhagic stroke, ischemic heart disease, heart failure, myocarditis, arrhythmia, including cases of atrial fibrillation, atrial flutter, paroxysmal supraventricular tachycardia, ventricular tachycardia or ventricular fibrillation, acute myocardial infarction and intracardiac thrombus), and having a health condition increasing the risk of cardiovascular events (obesity – Body Mass Index ≥ 30, hypertension, type 1 or 2 diabetes mellitus, chronic kidney disease or dyslipidemia), before the SARS-CoV-2 test. Additionally, we created a variable indicating whether the individual received a partial or complete vaccination scheme or was unvaccinated before the SARS-CoV-2 test.

Data analysis

The dataset underwent thorough validation procedures to ensure data integrity, encompassing checks for errors and inconsistencies. After validation, participant's sociodemographic and clinical baseline characteristics were described using absolute and relative frequencies. Our outcome of interest was cardiovascular events in the post-acute phase of COVID-19. Thus, we excluded individuals who experienced an event during hospitalisation or within 30 days after a positive SARS-CoV-2 test [17, 18, 32].

We estimated person-years for each individual included in the study, and the incidence rate was calculated with the respective 95% confidence interval (95%CI) for each event and at least one cardiovascular event. Cardiovascular events that occurred less than five times were not analysed individually but were considered for the combined outcome. We also estimated the incidence rate and respective 95%CI according to disease severity, cardiovascular risk, and vaccination status.

Follow-up started 30 days after the positive SARS-CoV-2 test until the date of the first cardiovascular event, the date of death or the date of last contact with the health services, whichever came first. Individuals who did not have any events were censured at their last contact or death. A univariable Cox proportional hazard regression was performed for each variable of interest. A multivariable Cox proportional hazard regression was then fitted, considering all the assessed variables. Heterogeneity between hospitals was accounted for by adding random effects in the model. We estimated crude and

adjusted hazard ratios and the corresponding 95% confidence interval for each variable.

Some hospitals are unable to access deceased individuals' Clinical Registry Records (RSE). In these situations, the registers wrote a note in the free text field. The main analysis included all individuals. We also performed a sensitivity analysis to assess the impact of incomplete records, excluding these individuals.

The analysis was performed using R, version 4.2.2 [33].

Results

Each hospital identified potential participants in their databases, resulting in a total of 2,200 participants. We excluded 284 participants who were not eligible. Of the eligible participants, we excluded 48 after data validation and 65 who had missing event dates or had an event before discharge or within 30 days after the SARS-CoV-2 test. The final sample included 1,803 participants (Fig. 1).

Table 1 presents the characteristics of the sample. Overall, more men (52.6%), with a mean age of 68, with at least one risk factor for cardiovascular disease (83%), and who had already received at least one dose of the vaccine against COVID-19 (83%), were included in the analysis. Only 25 children and adolescents were included in the sample, corresponding to 1.4%. Considering the low proportion of minors, these were not excluded from the analysis. Information about smoking, drinking habits and the practice of physical exercise was missing for more than half of the sample. Participants were followed for an average of 16 months. Regarding the severity of the hospitalisation, 23.9% had mild disease, without the need for supplementary oxygen, 60.6% had moderate disease, and the remaining 15.5% had severe disease, needing ventilation or ICU admission.

Regarding cardiovascular events, 143 participants experienced at least one cardiovascular event (7.9%), with heart failure being the most frequent (4%). The frequency

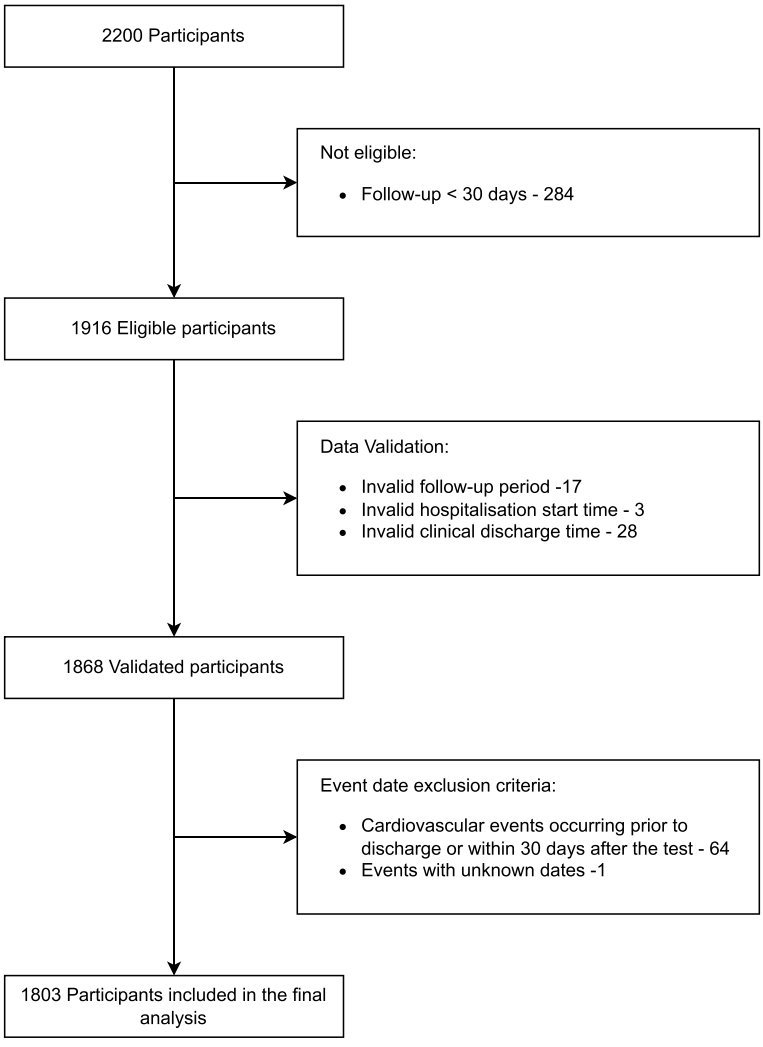

Fig. 1 Flowchart of the study indicating inclusion/exclusion criteria

Table 1 Sociodemographic and clinical baseline characterisation of the patients

Variable	N (%)
Sex	
Men	948 (52.6%)
Women	855 (47.4%)
Age	
Mean (Range)	68.2 (5.0, 99.0)
Median (IQR)	71.0 (58.0, 81.0)
Missing	3.00 (0.2%)
Death after hospital discharge	
No	1,568 (87%)
Yes	224 (12.4%)
Missing	11 (0.6%)
Follow-up duration (in months)	
Mean (Range)	16.3 (1.0, 33.3)
Median (IQR)	18.4 (10.8, 22.1)
Smoking status	
Smoker	69 (3.8%)
Former	209 (11.6%)
Never	551 (30.6%)
Unknown	974 (54.0%)
Drinking status	
No	450 (25%)
Yes	123 (6.8%)
Unknown	1,230 (68.2%)
Physical exercise	
No	377 (20.9%)
Yes	30 (1.7%)
Unknown	1,396 (77.4%)
Hypertension (yes)	1,172 (65.0%)
Hemorrhagic/ischemic stroke (yes)	180 (10%)
Ischemic heart disease (yes)	120 (6.7%)
Heart failure (yes)	312 (17.3%)
Myocarditis (yes)	3 (0.2%)
Atrial fibrillation (yes)	220 (12.2%)
Other arrhythmias (yes)	73 (4.0%)
Acute myocardial infarction (yes)	66 (3.7%)
Intracardiac thrombus (yes)	4 (0.2%)
Obesity (BMI ≥ 30) (yes)	467 (25.9%)
Type 1 or 2 diabetes mellitus (yes)	557 (30.9%)
Dyslipidemia (yes)	923 (51.2%)
Chronic kidney disease (yes)	172 (9.5%)
History of deep vein thrombosis (yes)	32 (1.8%)
Cardiovascular risk factor (≥ 1)	1,496 (83%)
COVID-19 vaccine	
Unvaccinated	306 (17%)
Partially vaccinated	1,266 (70.2%)
Completely vaccinated	231 (12.8%)
Severity of the episode	
Mild	431 (23.9%)
Moderate	1,093 (60.6%)
Severe	279 (15.5%)

of each cardiovascular event can be seen in Additional File 1.

The incidence of at least one cardiovascular event was 34.65 (95% CI: 29.20; 40.82) per 1,000 person-years. Overall, the incidence rate was similar across the cardiovascular events, varying between 30.83 (95% CI: 18.27; 48.73) for ischemic stroke and 46.43 (95% CI: 25.39; 77.91) for pulmonary thromboembolism per 1,000 person-years (Fig. 2).

We also estimated the incidence rate according to the severity of the COVID-19 episode, cardiovascular risk and vaccination status before the SARS-CoV-2 test (Fig. 3). The incidence rate, per 1,000 person-years, for at least one cardiovascular event varied from 1.50 (95% CI: 0.99; 2.19) for participants who had mild disease to 2.09 (95% CI: 1.69; 2.55) for those who had moderate disease. The difference according to the severity was minimal and not significant. Participants with pre-existing cardiovascular risk had a higher incidence rate than participants without cardiovascular risk (incidence rate (IR): 2.17, 95% CI: 1.82; 2.57 vs IR: 0.40, 95% CI: 0.13; 0.93). Participants who had received at least one dose of the COVID-19 vaccine had a lower incidence of cardiovascular events than unvaccinated participants (Fig. 3). We also estimated the incidence rate for each factor of the cardiovascular risk variable (Additional File 2). We found significant differences between participants with and without hypertension (IR: 2.33, 95% CI: 1.93; 2.80 vs. IR: 1.02, 95% CI: 0.67; 1.49), chronic kidney disease (IR: 3.35, 95% CI: 2.12; 5.03 vs. IR: 1.73, 95% CI: 1.44; 2.07) and atrial fibrillation (IR: 3.28, 95% CI: 2.18; 4.74 vs. IR: 1.70, 95% CI: 1.41; 20.4). Although not statistically significant, we also found differences between participants with and without ischemic heart disease (IR: 3.40, 95% CI: 1.98; 5.44 vs. IR: 1.77, 95% CI: 1.48; 2.11).

Table 2 presents the results of the univariable and multivariable Cox regression models. Participants with pre-existing cardiovascular risk were three times more likely to experience a cardiovascular event after COVID-19 (Adjusted Hazard Ratio (aHR): 3.76, 95% CI: 1.53; 9.24). We estimated a decrease of approximately 60% in the risk of a cardiovascular event for participants vaccinated before the SARS-CoV-2 test. We did not find an association between COVID-19 severity and the risk of a cardiovascular event. Results did not change when excluding cases where clinical staff could not assess the participant's Clinical Registry Records (Additional File 3).

Discussion

This study found that hospitalised COVID-19 patients had an incidence rate of 35 per 1,000 person-years for experiencing at least one cardiovascular event following the SARS-CoV-2 test. Additionally, having pre-existing cardiovascular risk was associated with an increased

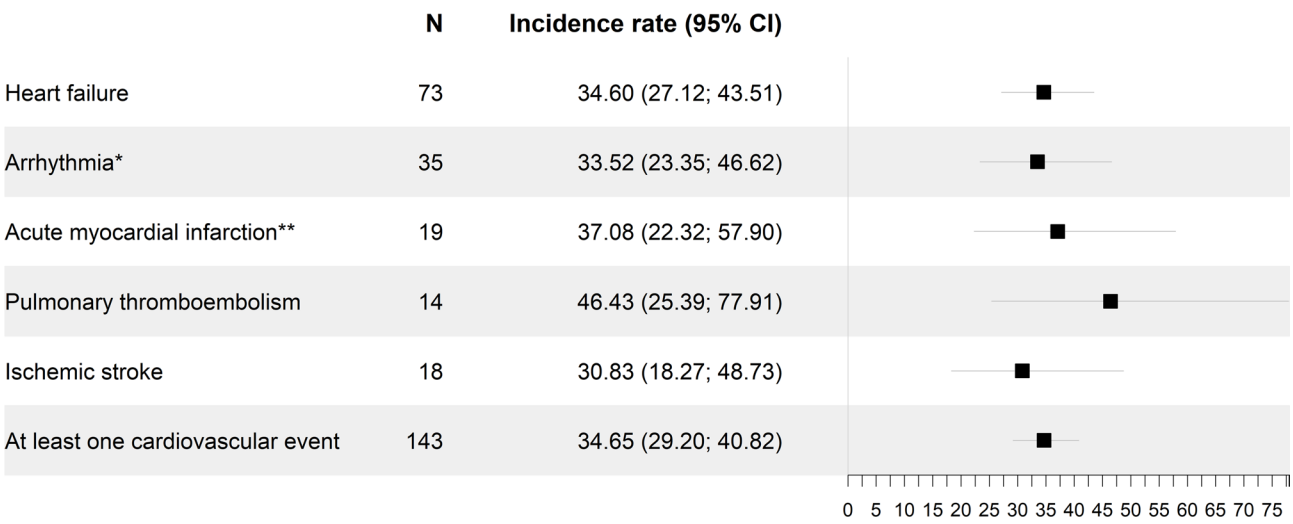

Fig. 2 Incidence rate of cardiovascular events per 1,000 person-years with the respective 95% confidence interval. N – number of events, *Arrhythmia including cases of atrial fibrillation, atrial flutter, paroxysmal supraventricular tachycardia, ventricular tachycardia or ventricular fibrillation, **Acute myocardial infarction with and without ST-elevation

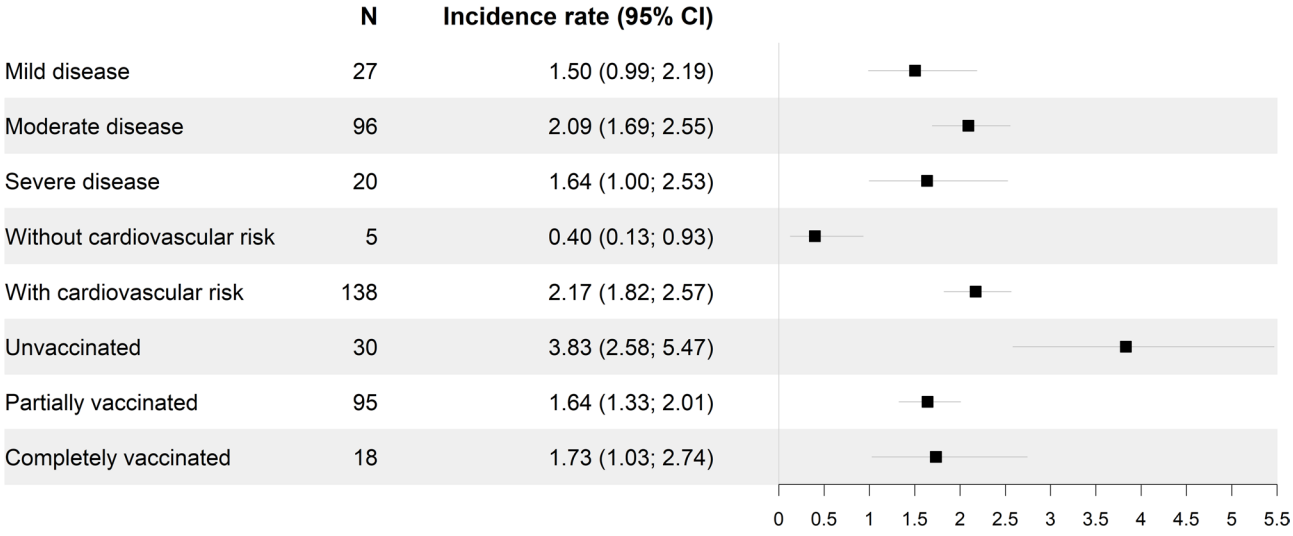

Fig. 3 Incidence rate of at least one cardiovascular event per 1,000 person-years with the respective 95% confidence interval according to the severity of the disease, cardiovascular risk and vaccination status before the SARS-CoV-2 test. N – number of events

incidence of cardiovascular events post-COVID-19, while vaccination against COVID-19 before the SARS-CoV-2 infection decreased the risk of cardiovascular events.

Several studies have explored the occurrence of cardiovascular events after COVID-19, although not all have provided estimates of their incidence rates. As mentioned, we estimated an incidence rate of 35 per 1,000 person-years for at least one cardiovascular event. However, this estimate varies considerably across studies, ranging from 2 to 126 per 1,000 person-years [34, 35]. Such heterogeneity persists even when considering prevalence estimates. We estimated a prevalence of 8% for at least one cardiovascular event, which also varied in the literature between 5 and 31% [34, 36]. Discrepancies in these figures may stem from differing composite outcomes evaluated across studies. While our study examined a spectrum of eight cardiovascular events, most studies focused on fewer events. However, even when considering individual cardiovascular events, we observed similar heterogeneity. Few studies have specifically examined incidence rates. To the best of our knowledge, estimates were only available for acute myocardial infarction and ischemic stroke [24, 35]. Our findings indicated an incidence of 31 per 1,000 person-years for ischemic stroke. Notably, this rate was substantially higher than previously reported, 4.5 per 100,000 person-years [35]. Considering individual events, we found

Table 2 Hazard ratios and 95% confidence intervals for cardiovascular events post-COVID-19

Variables	Crude		Adjusted*	
	HR	95% CI	HR	95% CI
Sex				
Men	—	—	—	—
Women	0.83	0.58, 1.19	0.78	0.53, 1.13
Age	**1.03**	**1.01, 1.05**	**1.02**	**1.01, 1.04**
Cardiovascular risk				
No	—	—	—	—
Yes	**5.52**	**2.28, 13.4**	**3.76**	**1.53, 9.24**
COVID-19 severity				
Mild	—	—	—	—
Moderate	1.44	0.89, 2.33	1.27	0.83, 1.92
Severe	1.10	0.53, 2.28	1.12	0.55, 2.31
Vaccination before SARS-CoV-2 test				
Unvaccinated	—	—	—	—
Partially vaccinated	**0.38**	**0.24, 0.60**	**0.44**	**0.30, 0.64**
Completely vaccinated	**0.40**	**0.21, 0.73**	**0.46**	**0.27, 0.80**

Significant values are represented in bold

HR Hazard Ratio, *CI* Confidence interval

*Adjusted by sex, age, cardiovascular risk, COVID-19 severity, and vaccination

a prevalence of 0.06% for myocarditis and 1% for ischemic stroke after COVID-19, which was higher than the 0.02% and 0.4% reported, respectively [24]. In contrast, we found a lower prevalence of pulmonary embolism and deep vein thrombosis than the pooled prevalence reported in a meta-analysis (0.8% vs 1.2% and 0.2% vs 2.3%, respectively) [24]. Heterogeneity in outcome definition, different study designs, sample sizes, and follow-up periods challenge comparison and explain some of the differences reported.

However, despite variations in reported estimates, numerous studies have consistently found a higher incidence and/or risk of cardiovascular events among individuals hospitalised with COVID-19 compared to control groups. For instance, individuals hospitalised with COVID-19 have been shown to have a higher risk of ischemic stroke compared to those hospitalised for other reasons [37]. A higher risk of ischemic stroke, arrhythmia, myocarditis, heart failure, and thromboembolic disorders was also reported in hospitalised COVID-19 patients compared to individuals without COVID-19 [17, 24, 38]. However, comparing these findings is challenging due to differences in control group selection. Individuals hospitalised with non-COVID-19 conditions may differ from those who test negative for SARS-CoV-2, potentially having a different clinical background with more comorbidities. Control groups should be carefully selected to estimate accurately the risk of cardiovascular events following COVID-19 hospitalisation and ensure comparability in background risk factors. Wang et al. matched controls to cases on several variables, such as age, gender, socioeconomic status, and comorbidities, among others [17]. Using matched control groups and considering potential confounders are essential to reducing bias and ensuring consistent comparisons. Due to resource limitations and ethical clearance, we were unable to use data from a control group. We compared the prevalences estimated in this study with those reported in Portugal. Our research indicates that the prevalence rates identified align closely with documented rates in the Portuguese population for certain cardiovascular events. We found a slightly higher prevalence of heart failure within our cohort compared to the general population (4% vs. 3%) [39]. Similar prevalences were found for deep vein thrombosis, and lower prevalences were observed for acute myocardial infarction (1.05% vs 3.2%), arrhythmia (1.94% vs 9%) and stroke (1% vs 1.6%) [40]. Thus, further studies in Portugal should explore whether cardiovascular events post-COVID-19 are indeed more frequent in individuals hospitalised with COVID-19.

Nonetheless, there are several possible biological mechanisms for the increased occurrence of adverse cardiovascular effects in post-COVID patients, which warrant research in this area. These patients might face increased cardiovascular issues due to COVID-19-induced inflammation damaging the cardiovascular system. Direct virus binding to ACE-2 receptors disrupts cardiac function, while inflammatory pathways exacerbate circulation problems and may potentially lead to plaque rupture [41–43]. Maladaptive responses, including heightened autoimmune reactions, persist beyond the acute phase. Delayed cardiovascular events may stem from lingering viral reservoirs in the heart, worsened by existing risk factors [44–47]. Although further research is required for confirmation, it is vital to understand who the individuals suffering from these cardiovascular events are.

Several factors have been identified as contributing to the increased risk of PCC. Our study, consistent with findings from Roubinian et al. [48] and Kim et al. [35], demonstrated a lower incidence of cardiovascular events among participants partially vaccinated against COVID-19 compared to unvaccinated individuals. Roubinian et al. [48] observed a higher risk and prevalence of post-hospital venous thromboembolism in unvaccinated COVID-19 patients compared to those vaccinated, while Kim et al. [35] reported higher rates of acute myocardial infarction and ischemic stroke in unvaccinated patients compared to fully vaccinated individuals [35, 48]. While the research investigating the impact of vaccination on severe cardiovascular events during the post-acute phase of COVID-19 remains limited, a recent review identified key conclusions regarding the interplay between vaccination status and post-acute COVID-19 symptoms [49]. These include the protective effect of pre-infection vaccination against developing post-acute COVID-19 symptoms, the potential benefit of post-infection vaccination

administered close to infection, and the reduction of symptoms and promotion of remission with post-infection and post-establishment of COVID-19 symptom vaccination. Nevertheless, further investigation is imperative to ascertain whether these observed impacts also extend to cardiovascular outcomes during the post-acute phase of COVID-19.

We also found that individuals with cardiovascular risk factors were at higher risk of cardiovascular events post-COVID-19, which is in line with results found in other studies [17, 18, 50]. A high proportion of our sample had pre-existing cardiovascular risk, which already increased their risk of a cardiovascular event regardless of COVID-19 hospitalisation. Nevertheless, other studies have adjusted or filtered by cardiovascular risk and still found an increased risk of post-COVID-19 cardiovascular events [17, 50]. Additionally, we also found an increased incidence of cardiovascular events post-COVID-19 among individuals with hypertension, chronic kidney disease, atrial fibrillation, and ischemic heart disease. Thus, these findings underscore the importance of identifying and closely monitoring individuals with pre-existing cardiovascular conditions, as they are at higher risk of experiencing cardiovascular events following COVID-19 infection.

We did not find significant differences in the incidence rate of experiencing at least one cardiovascular event based on the severity of hospitalisation. Xie et al. [18] obtained different results, showing that the risk of developing cardiovascular complications post-COVID infection increased gradually according to the care setting, during the acute phase (non-hospitalised, hospitalised, and admitted to intensive care). However, Lee et al. [37] did not find differences between individuals hospitalised with COVID-19 and individuals hospitalised with non-COVID-19. Therefore, although studies indicate differences in PCC prevalence between hospitalised and non-hospitalised patients [21, 24, 38, 51], it is essential to explore the effect of severity further and understand whether this phenomenon is characteristic of COVID-19 infection or respiratory infections in general.

Our study has some limitations. Firstly, we extracted information about several cardiovascular events and synthesised them into a composite outcome representing at least one cardiovascular event. Despite estimating the incidence of each cardiovascular event, the use of diverse composite outcomes in the literature challenges comparison and interpretation. Additionally, our approach to identifying cardiovascular events relied on data extracted from clinical processes rather than solely on ICD-10 codes. While we provided a manual to guide clinical staff in data extraction, describing how each question should be answered, the involvement of different hospitals and medical staff introduced potential subjectivity into the process, thereby impacting the consistency and reliability of our analysis. Additionally, information bias might also be present. Although medical staff involved in data collection received training and had a data collection manual available, they had access to the entire patient clinical record, which might unconsciously influence how they recorded or interpreted the data due to prior knowledge of the patient's history, conditions, or risk factors. Furthermore, our analysis only considered the information available in the clinical records, potentially leading to underestimating cardiovascular event incidences. This could occur if clinicians did not prioritise documenting cardiovascular events or if patients failed to report relevant clinical information, disregarding certain health conditions or events. The lack of a clear control group is a major limitation, as we are unable to ascertain whether the incidence of cardiovascular events is higher than the one reported by the general population or individuals hospitalised with other respiratory conditions. Similarly, we examined cardiovascular events following SARS-CoV-2 testing, but we cannot definitively attribute causality to COVID-19, as these events may have occurred independently. Additionally, individuals in our cohort were hospitalised at the beginning of the pandemic, when vaccines might not have been widely available for everyone. Moreover, studies have shown that vaccination after infection also decreased the odds of PCC. Additionally, with the appearance of self-testing, we cannot guarantee that individuals were COVID-19 free during follow-up, which might bias our estimates. Thus, caution should be used when interpreting our results.

Considerable attention has been focused on addressing the challenges individuals face with PCC. We extracted data from clinical records across major hospitals nationwide to assess the incidence and risk factors associated with cardiovascular outcomes for up to 16 months following a positive SARS-CoV-2 test among hospitalised individuals. Due to the heterogeneity in incidence rates of cardiovascular events post-COVID-19, it is crucial to design comparable studies and standardise outcomes for effective comparison. Furthermore, to thoroughly evaluate the impact of COVID-19 infection on cardiovascular complications, there is an urgent need for updated and broader studies examining the incidence of cardiovascular diseases. Current studies should delve deeper into the incidence rates of cardiovascular events by considering various groups and employing matching techniques to mitigate bias. Despite the need for additional research to fully comprehend the complexities of post-COVID cardiovascular events, our study reinforces the existing evidence indicating a heightened risk for individuals hospitalised with COVID-19. The risk of cardiovascular events post-COVID-19 was still evident, even with a longer follow-up (median of 18 months vs 12 months

[17, 18, 20]). This risk is particularly accentuated in those with pre-existing cardiovascular conditions, reinforcing the need to identify individuals at current risk of cardiovascular events post-COVID-19. In particular, we found a higher incidence rate for those with hypertension, chronic kidney disease, and atrial fibrillation, diseases which are not usually under study. Given these findings, it is imperative for health systems to proactively organise and implement screening protocols for individuals who were hospitalised with COVID-19 and with pre-existing cardiovascular risk factors. By identifying and closely monitoring these patients, healthcare providers can intervene early to mitigate the risk of post-cardiovascular events. This proactive approach can significantly improve patient outcomes and reduce the burden on healthcare resources by preventing or minimising the severity of cardiovascular complications associated with COVID-19.

Conclusion

Our findings indicate an increased incidence of cardiovascular events in individuals hospitalised with COVID-19. Individuals with pre-existing cardiovascular conditions are particularly at risk. We also found that vaccination against COVID-19 before infection lowers the risk of cardiovascular complications. This highlights the importance of proactive cardiovascular monitoring and strong vaccination campaigns, especially for those at increased risk. It's vital for both healthcare providers and individuals who have recovered from COVID-19 to remain vigilant about cardiovascular health over the long term.

Abbreviations

WHO	World Health Organisation
COVID-19	Coronavirus disease 2019
PCC	Post-COVID Condition
MRI	Magnetic Resonance Imaging
ICU	Intensive Care Unit
ICD	International Classification of Diseases
CI	Confidence interval
RSE	Clinical Registry Records
IR	Incidence rate
aHR	Adjusted Hazard Rat

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12879-025-11762-0.

Supplementary Material 1.

Supplementary Material 2.

Supplementary Material 3.

Supplementary Material 4.

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Authors' contributions

PS, MJL, LS, JP, IS, LEG, AI, GA, SN, MLB and AL contributed to the design of this study. MJL, LS, JP, ARR, AH, IS, LEG, RM, AI, GA, SN and MLB contributed to data collection. Data validation and statistical analysis were performed by PS, with support from CR, JS and AL. PS, CR, JS, JP, ARR and AH produced the first draft of the manuscript. All authors reviewed, edited, and approved the final version.

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Data availability

The datasets generated and analysed during the current study are currently embargoed but are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate

The study design and protocol are compliant with fundamental research ethics norms and documents and have been reviewed and approved by the competent Ethical Review Boards. The Ethics Committee for Health from each participating hospital approved the study protocol (Additional File 4). All hospitals waived the requirement for informed consent as data was completely anonymised.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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